Acute aortic occlusion – Factors that influence outcome

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Purpose: **The purpose of this study was to report our experience in the management of acute aortic occlusion and to analyze factors that influenced the outcome.**
Methods: **This was a retrospective analysis of 48 patients with acute aortic occlusion treated over a 19-year period. Presentation included limb ischemia in 34, acute abdomen in four, spinal cord compression-like symptoms in eight, and sudden onset of hypertension in two patients. Thrombosis was the cause of acute aortic occlusion in 44, and embolus in four patients. Acute thrombosis was associated with underlying atherosclerotic occlusive disease in 36 patients. In these, thrombosis was due to low-flow state caused by cardiac dysfunction or severe volume depletion. Thrombosed aneurysms caused aortic occlusion in two patients. Hypercoagulable state caused thrombosis of relatively normal aorta in six patients. Angiography in 39 patients revealed occlusion to be juxtarenal or infrarenal in 37 and suprarenal in two. Left ventricular function (LVF) was assessed in 42 patients. Circulation was restored in 45 (aortofemoral bypass in 22, axillofemoral bypass in 12, and thromboembolectomy in 11). This was not feasible in three patients. Additional surgical procedures were required in 29 patients (64%).**
Results: **The overall mortality rate was 52% (25 of 48). Of the 20 patients with severely compromised LVF, 17 died (85%). In contrast, only five (23%) deaths occurred among 22 with good LVF. Among 29 patients who required additional operations, 18 died (62%). All four patients with embolic occlusion survived. Patients with normal LVF but hypercoagulable state had dismal outcome – only one of the six survived.**
Conclusions: **Acute aortic occlusion is infrequent. Presentation may be varied, thus delaying diagnosis. Poor LVF, thrombosis of arteries below the inguinal ligament or of visceral arteries, and "hypercoagulable state" portend ominous prognosis. (J VASC SURG 1995;21:567-75.)**

Occurrence of acute occlusion of aorta either because of thrombosis or embolism is an infrequent clinical entity. Except for a recent report by Dossa et al.,[1] many of the large reports of aortic occlusion mainly describe chronic occlusion of the abdominal aorta (Table I).[2-7] Chronic aortic occlusion is clearly a different disease entity, and principles of diagnosis and management do not apply to the acute aortic occlusion. There have been isolated reports of acute aortic occlusion describing unusual causes and atypical presentation.[8-12] Even when diagnosed promptly, management requires thorough understanding of the underlying factors that contribute to the development of occlusion. Revascularization by itself does not ensure successful outcome in these patients in whom this may represent either end-stage cardiovascular dysfunction or poorly understood derangement of coagulation homeostasis.[13-15] Previous reports on acute aortic occlusion have alluded to the difficulties in diagnosis and challenges in management leading to poor outcome (Table II).[15-24] We report a retrospective review of 48 patients with acute aortic occlusion and propose an algorithm for management (Fig. 1). Aortic occlusion caused by trauma, aortic dissection, and intraaortic balloon pump were not included in this study. Based on a Medline search from 1966 to 1994, this represents the largest series of acute aortic occlusion in the English literature.

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Presented at the Eighth Annual Meeting of the Eastern Vascular Society, Montréal, Québec, Canada, May 12-15, 1994.
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0741-5214/95/$3.00 + 0 **24/6/62357**

PATIENTS AND METHODS

Medical records of 48 consecutive patients treated for acute aortic occlusion caused by either thrombosis or embolism were reviewed. There were a total of 48

Table I. Large series reporting aortic occlusion

Author	*Period (yrs.)*	*Chronic occlusion (No.)*	*Acute occlusion (No.)*
Dossa[1]	40	0	46
Starrett[2]	23	106	0
Liddicoat[3]	6	64	0
Corson[4]	16	63	5
McCullough[5]	17	38	0
Deriu[6]	1	30	0
Ketonen[7]	15	55	0

patients over a 19-year period (1974 to 1993). Twenty-six were women, and 22 were men. Age of the patients ranged from 35 to 85 years.

Clinical presentation. Presentation included acute limb ischemia in 34, neurologic deficit simulating acute spinal cord compression (paresthesia, paresis, paralysis) in eight, abdominal pain in four, and acute hypertensive crisis in two patients, both of whom had extension of thrombus into the renal artery. In the acute ischemia group, 18 had paresthesias, but the ischemic symptoms were more pronounced. Twenty-eight patients had significant coronary artery disease, and 29 had a history of hypertension. Duration of ischemia was less than 8 hours in 29 patients and more than 8 hours in 14 at the time of presentation. The exact onset time of symptoms could not be determined in five patients. Diagnosis of acute aortic occlusion was delayed from 10 hours to more than 3 days in all 14 patients with clinical presentation other than limb ischemia. In one patient myelography was performed before the diagnosis of acute aortic occlusion was made.

Cause. Except for four patients who had embolic occlusion, all the rest had acute aortic thrombosis. In all four patients with embolic occlusion, the aorta was normal, and emboli originated in the heart. Two of these had left ventricular thrombus diagnosed by echocardiography. In one patient diagnosis of myxoma was made on clinical and histologic studies. In this patient myxoma was presumed to have originated from the heart even though conventional and transesophageal echocardiography results were normal. The fourth patient had documented valvular heart disease, which was believed to be the source of aortic embolus. Among 44 patients with acute aortic thrombosis, 36 patients had underlying atherosclerotic occlusive disease, two had small abdominal aortic aneurysms (<5 cm). In the remaining six patients with acute thrombosis of normal aorta, the diagnosis of "hypercoagulable state" was eventually confirmed by hematologic studies. Cause of sudden thrombosis in 36 patients with underlying atherosclerotic occlusive disease was precipitated by low flow state (20 patients) caused by dehydration (volume depletion because of poor intake, aggressive diuresis, or third space fluid loss) or cardiac decompensation (16 patients) caused by congestive heart failure, acute myocardial infarction, or sudden intractable arrhythmias.

In the subgroup of patients with "hypercoagulable state," diagnosis of heparin-associated thrombosis in three, protein C deficiency in one, and anticardiolipin antibody in one was made. The definitive cause of "hypercoagulable state" could not be made despite hematologic, immunologic, and histochemical studies in the sixth patient.

Management. Preoperative angiograms were obtained in 39 patients. In the remaining nine, angiography was not performed because the diagnosis of aortic occlusion was obvious, and ischemia was advanced at the time of initial presentation. Angiograms revealed occlusion to be juxtarenal or infrarenal in 37 and suprarenal in two patients. In these two patients, aortic dissection was suspected because of absent femoral pulses, lack of urine output, and back pain. The angiogram helped to make the correct diagnosis of suprarenal aortic occlusion. There were no cases of visceral emboli in this series. When aortic occlusion was seen on angiography, femoral arteries were almost always visualized (35 patients). To limit the contrast load in this group of physiologically fragile patients, no specific attempt was made to visualize arteries distal to the femoral arteries once the diagnosis of aortic occlusion was made.

After the diagnosis of aortic occlusion was established, patients were immediately given heparin while measures were taken to improve their state of hydration and cardiovascular function. Flow-directed pulmonary artery catheters and radial lines were placed to obtain hemodynamic data in 42 patients. Left ventricular function was assessed by plotting the left ventricular stroke work (LVSW) against pulmonary capillary wedge pressure (PCWP). Pharmacologic therapy was initiated on the basis of the position of the left ventricular function (LVF) in relation to the Sarnoff ventricular function curve.[14] This guided the operative approach to revascularization. Extraanatomic bypass was preferred in patients with

Table II. Previous reports of acute aortic occlusion: types of operations and mortality rates

Author	*No. of patients*	*Years*	*Management*	*Mortality (%)*
Bell[15]	6	10	Aortic opn	100
Danto[16]	9	9	Aortic opn	25
Johnson[17]	7	–	Aortic opn	53
Drager[18]	6	3	Axfem	16
Kornmesser[19]	22	10	Aortic opn/Embolectomy	41
Litooy[20]	18	8	Aortic opn/Embolectomy	50
Pietri[21]	10	12	Aortic opn	60
Webb[22]	10	10	Axfem/Aortic opn	50
Ross[23]	26	8	Embolectomy	30
Bradbury[24]	14	9	Aortic opn	14
Dossa[1]	46	40	Aortic opn/Axfem/Embolectomy	35
Present report	48	19	Aortic opn/Axfem/Embolectomy	52

opn, Operation; *Axfem*, Axillofemoral bypass.

compromised LVF (LVSW less than 40 gm/m/m^2 with PCWP of 20 mm Hg or greater). Restoration of circulation was achieved in 45 patients; 22 aortofemoral bypasses, 12 axillofemoral bypasses, and 11 thromboembolectomies (10 transfemoral, 1 aortic) were performed. Diffuse atherosclerotic disease mandated primary, bilateral, high lower-limb amputation in one patient. Moribund state at presentation precluded any intervention in two patients. Simultaneous, sequential, and additional vascular and extravascular procedures were required in 29 patients. These included infrainguinal or infragenicular vascular procedures (n = 19), bowel resections (n = 3), fasciotomy for compartment syndrome (n = 6), and major limb amputations (n = 9).

RESULTS

There were 25 deaths (52%). As shown in Table III, 20 patients had severely compromised LVF, 17 of whom died (85%) despite surgical correction of acute aortic occlusion. The compromised cardiac function in this subgroup did not respond to pharmacologic therapy. In contrast, there were only five deaths (23%) among 22 patients who had adequate LVF at the time of presentation or whose LVF improved in the perioperative period. Improvement was defined as LVSW greater than 40 gm/m/m^2 associated with reduction in PCWP to 20 mm Hg or lower. Among 29 patients who required additional procedures for limb or visceral ischemia, 18 died (62%). Of nine patients who required major limb amputations, seven died (78%). Advance distal vessel disease in four patients, hypercoagulable state in three and irreversible ischemia at the time of initial presentation in two were the reasons for amputation. Hence, severity of ischemia rather than duration appeared to be the influencing factor. Of the 36 patients with primary atherosclerotic occlusive disease, 20 died (55%). The subset of patients with normal heart and arteries who had thrombosis of their aorta because of clotting diathesis had dismal outcome. Only one of the six survived in this group (83% mortality rate), and three of the six had limb loss, including the surviving patient. Both patients with suprarenal aortic occlusion (atherosclerotic disease in one and hypercoagulability in the other) died. All eight patients who were admitted with symptoms of spinal cord involvement had complete resolution of neurologic symptoms after restoration of circulation. All four patients with embolic occlusion survived.

DISCUSSION

Acute aortic occlusion is an uncommon occurrence judging by the experience of various authors who have reported small series seen over extended time periods.[14-24] The incidence, hence, must be low given that diagnostic techniques such as computed tomography and ultrasonography have become routine in treatment of patients with vascular disease.

Contrary to what might be expected, not all patients with sudden occlusion of abdominal aorta are admitted with symptoms of severe ischemia. This was amply demonstrated in our series in which 14 of the 48 patients were diagnosed with symptoms other than lower extremity ischemia. Eight patients were initially believed to have spinal cord disease (either compression or infarction). The symptoms in this group ranged from sudden onset of lower extremity paresthesia to frank paraplegia. Others have reported similar presentation of acute aortic occlusion.[7,11,12,25,26] Experimental studies have shown that the neurologic manifestations of acute aortic occlusion do not imply spinal cord infarction but severe ischemic neuropathy.[27] All our patients with symptoms of spinal cord dysfunction had total resolution of symptoms after restoration of circulation, which further supports the reversible nature of the neuro-

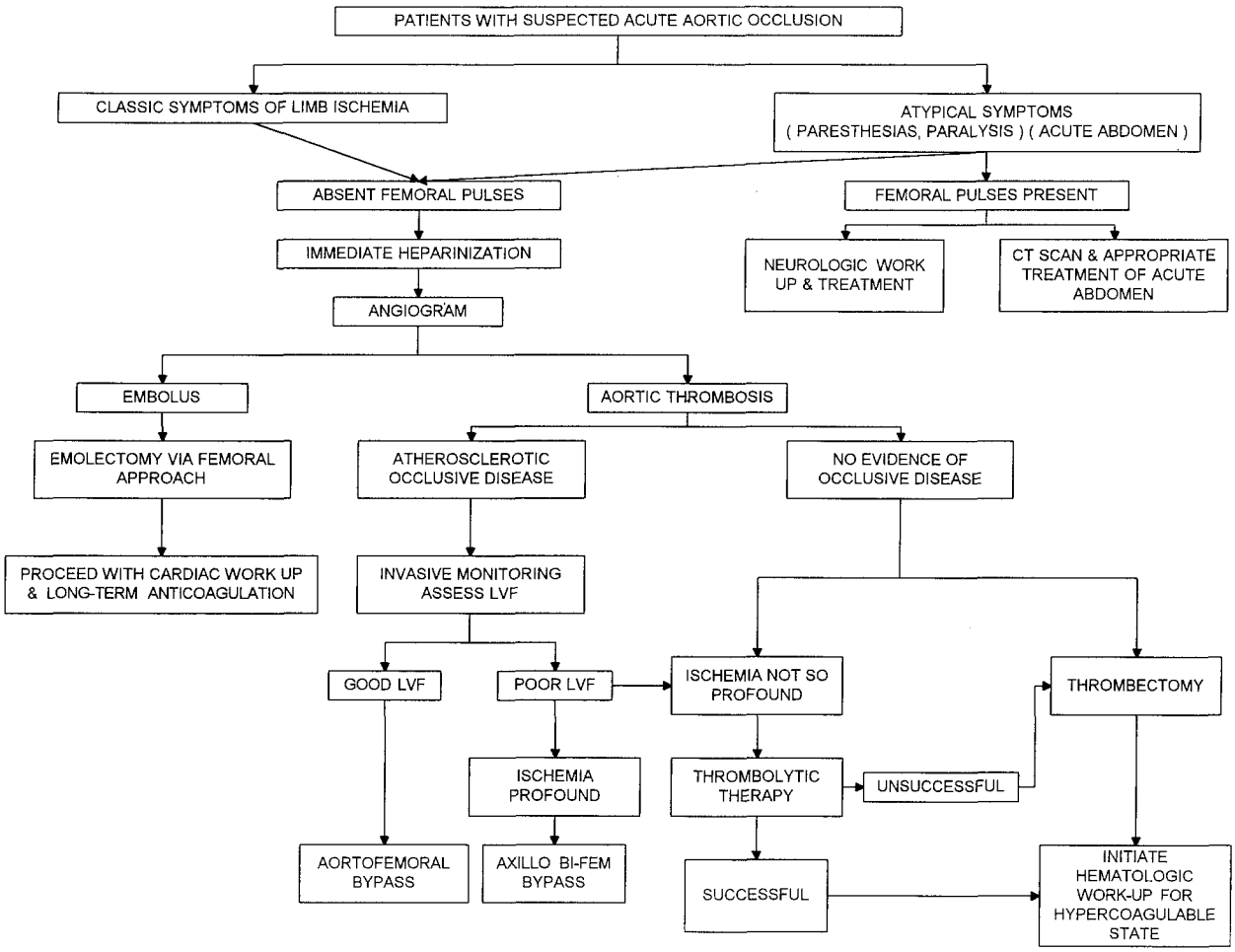

Fig. 1. Proposed management strategy for patients with acute aortic occlusion.

logic deficits. However, in a 40-year retrospective review of acute aortic occlusion by Dossa et al.,[1] presence of moderate to severe motor or sensory deficit was shown to correlate with death. The neurologic symptoms in these patients with aortic occlusion may mislead the treating physician and delay the diagnosis. Major vascular occlusion should be considered in the differential diagnosis of any patient diagnosed with sudden neurologic deficit. When patients are sent for imaging studies such as computed tomography or magnetic resonance imaging in pursuit of spinal cord disease, the aorta should be assessed when spinal cord study results are negative. Moore et al.[11] have suggested that this may obviate the need for further invasive studies such as angiography and will expedite correct diagnosis and appropriate management. However, angiography should generally be performed in all instances unless the patient's clinical condition precludes it. In our experience, besides confirming the diagnosis, angiography played a vital role in assessing and planning management. Angiography demonstrated extension of aortic thrombus to the renal artery in two patients diagnosed with acute hypertension. Fragmentation of embolus to vessels distal to the aortic bifurcation was documented by preoperative angiography in one patient, thus guiding appropriate operative procedure in addition to aortic embolectomy. Angiography will also help in delineating the status of visceral circulation, particularly in those patients admitted with symptoms of acute abdomen and absent femoral pulses.

In patients diagnosed with symptoms of lower limb ischemia, the degree of manifestations ranged from mild (cool extremities) to severe (mottled, cyanotic extremities, compartment syndrome, myonecrosis). The threshold for tolerance to sudden aortic occlusion was very low in some patients who had rapid progression to compartment syndrome and kidney failure, which complicated the management. Because of the variability of clinical presentation and the difficulty in establishing the absolute time of onset of aortic occlusion, statistical analysis of factors predictive of low tolerance to ischemia was not

possible. However, patients with hypercoagulable state appeared to have the lowest threshold for sudden interruption of circulation distal to the aorta. This could be explained by thrombosis of microcirculation or the inability of collateral vessels to compensate for the acute aortic occlusion (because these patients had no preexisting occlusive disease).[6]

Only four patients in this series had embolic occlusion. Most reports of large series of embolic occlusion are from countries where rheumatic heart disease is still prevalent.[28] When occlusion of the aorta is caused by embolism, emboli in the arteries distal to the aorta should be ruled out after aortic embolectomy is performed. One of our patients showed significant improvement of her limb ischemia after aortic embolectomy. However, lack of palpable pedal pulse prompted repeat angiography, which revealed embolic occlusion of the popliteal artery, which was promptly treated. Preoperative angiography may not always show distal emboli because of poor visualization of distal vessels when there is major aortic occlusion. Hence, adequacy of embolectomy should be confirmed by intraoperative angiography if pedal pulses are not present after aortic embolectomy. Whereas most patients with embolic occlusion of the aorta may be treated with retrograde transfemoral embolectomy, one of our patients required direct aortotomy to retrieve an embolus that was firmly wedged in the aortic bifurcation. All four patients survived even though high mortality rates have been reported in the literature for embolic occlusion of the aorta.[20,23]

Aortic occlusion was due to thrombosis in 44 patients. Occlusive vascular disease was the underlying cause in 36, which was limited to the aorta in eight but involved the iliac and femoral arteries and distal vessels in 28 patients. When low flow states occur, the development of acute thrombosis in patients with preexisting chronic vascular disease has been noted by others.[14-16,20-22,24]

Abrupt thrombosis of aneurysms was the cause of acute aortic occlusion in two patients, both of whom survived without sequelae. Thrombosis of small aneurysms, the disastrous consequences of such thrombosis, and mortality rates in these patients approaching those of ruptured aneurysms have been reported.[15-17] When patients are admitted with acute aortic occlusion, thrombosis of aortic aneurysms should be considered in the differential diagnosis.

Abnormal clotting diathesis has only recently been described as a cause of acute aortic thrombosis.[13,29] Detailed hematologic evaluation should be performed in any patient diagnosed with acute aortic thrombosis who is free of heart or vascular occlusive disease.

Table III. Acute aortic occlusion: factors affecting outcome

Factors	*No. of patients*	*Mortality (%)*
Good LVF	22	5 (23)
Failing ventricle	20	17 (85)
Additional procedures	29	18 (62)
Limb amputations	9	7 (78)
Hypercoagulable state	6	5 (83)
Embolic occlusions	4	0 (0)

LVF, Left ventricular function.

Preoperative management, including administration of heparin, hydration, and optimization of cardiac function, are as important as the operative intervention for successful outcome. We have previously reported the significance of such monitor-guided responses in the treatment of critically ill patients.[30] When the data obtained by invasive monitoring revealed suboptimal LVF, the operative approach was modified if feasible. Extraanatomic bypasses were performed preferentially. Drager et al.[18] have recommended the routine use of axillofemoral bypass in the treatment of patients with acute aortic occlusion. Extraanatomic bypasses have lower patency rates and, hence, may not be ideal for all patients. We believe that direct aortic reconstruction is preferable in those patients whose LVF is adequate or who have evidence of renal or mesenteric involvement associated with acute aortic thrombosis. Both femoral arteries should be exposed initially because femoral arteries are the site of distal anastomosis whether the inflow is chosen to be the aorta or axillary artery. Thrombectomy should be attempted by the femoral approach. This is the procedure of choice in those patients with acute aortic thrombosis or embolism and no evidence of preexisting occlusive disease. No patients in our series were treated by thrombolysis, although recent reports have proved both the safety and efficacy of thrombolytic therapy in infants and adults.[31,32] This may be preferable therapy in patients with acute aortic thrombosis caused by "hypercoagulable state" because this subset of patients fared poorly with surgical intervention despite normal arteries and good LVF.

There were 25 deaths among 48 patients, accounting for an overall mortality rate of 52%. Previous reports have highlighted the high mortality rate associated with acute aortic thrombosis (Table II). Seventeen of 20 patients (85%) with severely compromised LVF died, in contrast to five deaths among 22 (23%) patients with good LVF. It appears that acute thrombosis of the aorta represented the terminal event in the subgroup of patients with

advanced vascular disease and failing heart. Retrospectively, hypercoagulable state, atherosclerotic disease extending beyond the aortoiliac segment, and intractable heart failure all appeared to be clues to the necessity for additional operative interventions.

Acute aortic occlusion is an infrequent occurrence. Symptoms other than limb ischemia at the time of presentation may mislead the treating physician and delay the diagnosis in as much as one third of patients. Once the diagnosis is made, therapy should be based on sound clinical judgment supported by hemodynamic parameters. The management spectrum ranges from simple thromboembolectomy, extraanatomic bypass, and aortic reconstruction to thrombolytic therapy. Based on our experience and a review of the literature spanning three decades, we propose a systematic approach to the evaluation and treatment of these critically ill patients (Fig. 1). The disappointing outcome in patients with "hypercoagulable state" underscores the need for further research into the factors that derange coagulation homeostasis.

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Submitted May 31, 1994; accepted Nov. 22, 1994.

DISCUSSION

Dr. William C. Mackey (Boston, Mass.). The authors have presented a large, skillfully treated, and carefully analyzed series of 48 patients with acute embolic or thrombotic abdominal aortic occlusion. Notable is their finding that nearly one third of these patients are diagnosed with symptoms other than acute limb ischemia. The eight patients diagnosed with neurologic symptoms are especially interesting and should increase our awareness of this clinical scenario. Also notable is the lethality of this condition. Even with state-of-the-art resuscitation, monitoring, hemodynamic support, and surgical management, more than half of their patients in their series died. Correlates of death in their patients were poor ventricular function; the extent of ischemia, vascular disease, or end-organ damage at presentation as measured by the need for ancillary procedures; and the presence of hypercoagulability.

Why did you choose not to include aortic occlusion caused by an intraaortic balloon pump (IABP) or dissection? The comorbidity factors, management guidelines, and outcome in these patients should not be very different from those in the 48 patients you did analyze. How many IABP and dissection occlusions were there during your 18-year study period and what was their outcome?

Your series spans 18 years. How has your treatment of these patients changed over this period? Have your management changes resulted in improved survival rates?

From an economic and outcomes analysis viewpoint, these patients undoubtedly represent a disaster. Based on your current understanding of these patients, can withholding care be justified? Can patients whose likelihood of survival is nil be identified before therapy?

Finally, do you have any follow-up data on your survivors? If the acute survivors have very poor long-term survival rates, intervention with its extensive resource allocation and 52% acute mortality rate, may be difficult to justify. On the other hand, if long-term survival is excellent, as in patients diagnosed with chronic limb ischemia because of aortoiliac disease, intervention can be more easily defended.

Dr. Sateesh C. Babu. Regarding use of the IABP, we treated three or four patients. We did not include them because this condition was partly iatrogenic and partly due to preexisting disease. The outcome in this subset was poor because of delay in therapy because of these cardiac complications.

We did not have any patients with trauma, although there have been written reports of aortic occlusion caused by trauma, so that was excluded.

Regarding improvement of results in these 18 years, our results have not shown improvement. The surgical principles and the physiologic monitoring have not changed in our hands during this time span.

Whether we should select which patients should undergo surgical therapy is a difficult dilemma. For example, the last patient we did not operate on had extensive ischemia from the umbilicus down to the toes, which we call premortem rigor mortis. Our decision not to operate was a difficult one and hard to extrapolate to general guidelines.

The real reason our results have not improved is due to poor control of thrombogenesis. The hypercoagulable state was the major challenge for us. These patients were frequently young patients with normal-looking arteries; however, within a few hours after surgical thrombectomy, they were thrombosed again. I do not think that we have the full answer of how to treat these patients.

Regarding follow-up, I have to say those patients whom we successfully operated on are all doing well. In fact, we are still monitoring some of them, although I do not have the exact number to give you. Those patients on whom we operated are doing well. Should we perform axillofemoral bypass routinely in these people? Some patients had active peritonitis as a presenting picture, and we had to explore the abdomen to make sure that they did not have associated ischemic bowel or some other problems.

Dr. Enrico Ascer (Brooklyn, N.Y.). I share the same anxiety and frustration in treating these patients who are very sick; we try to do a quick thrombectomy or embolectomy and think that's enough. However, we have learned a couple of lessons the hard way over the years. One is that, after the embolectomy, we have to be sure that our inflow is sufficient, because many of these patients will have thrombosis the next day, and we think that it was an inadequate thrombectomy when actually the patients had underlying disease. So we routinely measure pressures in the femoral artery. The other lesson that we have learned, which has accounted for the good results over the last 3 or 4 years when we had 16 patients with only one death, is that we have been very aggressive about doing axillobifemoral bypasses after the first embolectomy. In the operating room, if the pressures are not good and our completion angiogram is not satisfactory, we perform axillobifemoral bypass. But we also learned that preoperative arteriograms are very helpful in evaluation of these patients, not only to confirm and try to delineate the outflow, but more importantly to examine the inflow vessels for an axillofemoral bypass. So these are lessons that have helped us over the last 3 or 4 years to improve our results.

Dr. Babu. I agree with you, particularly about defining the inflow when you perform axillofemoral bypass. I also have stressed that thrombectomy usually is uniformly unsuccessful if there is preexisting vascular disease. So we advocate some form of vascular reconstruction such as aortofemoral or axillofemoral bypass grafts. We did not discuss the selection of aortic reconstruction versus axillofemoral bypass based on an evaluation of ventricular function. For patients with poor ventricular function, one should expose both femoral arteries, try to do a thrombectomy, and, if it fails, proceed immediately to axillofemoral bypass in these patients.

Dr. Keith D. Calligaro (Philadelphia, Pa.). Can you assess the role of lytic treatment in these patients, assuming you do not have to do a laparotomy for other intraabdominal findings and assuming the legs are not that acutely ischemic where you think you might have a few hours.

My second question has to do with the hypercoagulable states you mentioned about the heparin. You state that the heparin was the cause of the arterial thrombosis. Why should the patients have been receiving heparin unless they were in the hospital for other reasons?

Dr. Babu. The hypercoagulable state was confirmed only after the fact. This was a patient who had a transient ischemic attack, was given heparin by the neurologist, and, within about 6 hours, presented with severe ischemia on the lower half of the body. We immediately tried to do a thrombectomy, administered more heparin, and were unsuccessful. Twenty-four hours later the hematologist confirmed that this patient had heparin-associated thrombosis. This patient exemplifies a major problem when we have a hypercoagulable state, we do not know this until after the fact. Therefore we use an algorithm in those patients diagnosed with sudden aortic thrombosis in whom we do not suspect a preexisting vascular disease, or who do not have heart disease. These patients may have a hypercoagulable state, and we may need to think of alternative treatments other than surgery.

With regard to your second point of thrombolytic therapy, none of our patients had thrombolytic therapy, although there are three or four cases in the literature of successful treatment of aortic thrombosis with thrombolytic therapy, and we have included it in our algorithm. Some patients who do not have ischemia when they are admitted may be candidates for thrombolytic therapy, particularly in the hypercoagulable group.

Dr. Jonathan P. Gertler (Boston, Mass.). We prospectively examined coagulation parameters in a variety of patients after vascular surgery. Routinely there is derangement in fibrinolytic function and coagulation function resulting in a procoagulant state. It turns into an uncertainty principle because you are measuring patients after surgery and therefore you may detect acute changes in coagulation that do not reflect the underlying state of the patient. Heparin-induced thrombocytopenia has been mentioned, and these patients must have been hospitalized. Are other patients in your hypercoagulable group also hospitalized before aortic occlusion?

Dr. Moshe Haimov (New York, N.Y.). These patients frequently present with severe spinal cord dysfunction. What is your experience about the reversibility of that dysfunction in the patients who survive? Would aortic reconstruction where you can open the lumbar circulation be better for a patient like this than axillobifemoral where you leave the cord ischemic?

Dr. Babu. It is interesting that the neurologic deficit was previously believed to be due to spinal cord ischemia in aortic thrombosis. However, this does not appear to be true. Several experimental studies have shown that the neurologic picture that we see in acute aortic infrarenal occlusion is different than the picture in thoracic aortic occlusion or dissection. The neurologic picture after infrarenal aortic occlusion is actually an intense ischemic neuropathy that is totally reversible. All eight patients in our series had reversible neurologic deficits. What we need to keep in mind is that these patients did not present with severe ischemia initially, but with neurologic symptoms. In fact, one or two of these patients even underwent myelography with the assumption that this was actually a spinal cord problem.

Dr. Syde A. Taheri (Buffalo, N.Y.). Heparin-associated thrombosis is an extremely rare condition, perhaps 3% of cases, and therefore we are dealing with positive antibody in probably 95% of our cases. If our patients don't respond to heparin, should we add some steroids to this treatment?

Dr. Babu. It is tough to say. As Dr. Gertler pointed out, the diagnosis of hypercoagulable state is not easy. This diagnosis sometimes takes 10 to 15 days to establish, with our rheumatologists and hematologists working together. Antiphospholipid antibody was confirmed in one patient, but it would be difficult to know beforehand, and I do not know whether the steroids will help just on the basis of suspicion. One of the patients in whom we were unable to determine the exact nature of hypercoagulable state was given azathioprim on the assumption that this was an immune problem, but he continued to have intravascular thrombosis.

Dr. Thomas S. Riles (New York, N.Y.). The group that is hypercoagulable is separate from the others, and I have little experience with it. My experience at the time we wrote our study was that the vast majority of these patients have some underlying heart problem, and that is usually what precipitates it. Either they had a myocardial infarction, they had the embolus from the heart, or they were overly treated by the cardiologists, which makes their condition very unstable at the time that you see them; this is why I think your options are somewhat limited.

Although you still recommend angiography, out of how many patients did the angiogram really give you useful information? If the patient is making good urine and has no pulses and has obviously ischemic limbs, you can figure out from that that the occlusion is in the infrarenal aorta. In fact, our experience has been that, if the angiogram is obtained above the occlusion, there is a high incidence of renal insufficiency because of their underlying condition and also that all of the dye is going into the renal arteries. Therefore we go straight to the operating room, cut down on the femoral arteries, pass the Fogarty catheters, and perform axillobifemoral grafting. Usually we like the sequence of diagnosis, angiography, and then surgery. However, this is one situation that has to be treated as an emergency, and the primary goal is to restore flow to the lower limbs as soon as possible.

Dr. Babu. I agree with you. When the diagnosis is certain, we probably do not need the angiogram. Whether

we do a thrombectomy or whether we are going to do an aortofemoral or axillofemoral bypass, the femoral arteries will be the common outflow tract, so I agree that the femoral arteries should be exposed first. However, the diagnosis was not always certain. Fourteen patients had the diagnosis much in doubt because their presentation was very unusual. With present day arterial digital techniques, very little dye is used, and, perhaps if you have the convenience of doing it quickly, you would still prefer to obtain the angiogram to know that you are not missing something. For example, if it was an aortic dissection, the whole management would be different.

Dr. Clark E. Williamson (Philadelphia, Pa.). I am interested in your patients who had hypercoagulable states. I had a patient who had an aortic occlusion the first night after aortobifemoral bypass who had no technical problems when we transected the aorta, had white thrombus sitting in the aortic stump, and later was documented to have heparin-induced thrombocytopenia. The only way we were able to save that patient was to obtain some ancrod and prevent clotting. We were able to save the patient's life at that point. So have you had any experience with this medication in this subgroup of patients with a hypercoagulable state?

Dr. Babu. That is very interesting information. We were not able to make the diagnosis so rapidly. We diagnosed this heparin-associated thrombosis later, by which time this patient had already lost a limb. If we have a high index of suspicion, we may use either thrombolytic therapy or ilioprosts.

Dr. John J. Ricotta (Buffalo, N.Y.). It is a little dangerous to say that these neurologic problems are always completely reversible.

How often did you perform fasciotomies in these patients? Do you have any suggestions about how to manage the legs? How do you treat these patients with heparin-induced thrombocytopenia? I treated several patients who ended up undergoing amputation.

Dr. Babu. I agree with you that there are some literature suggesting that paraplegia or the neurologic deficit has been irreversible in acute aortic occlusion. But in these eight patients in our series, all of them had reversible neurologic deficits. So the neurologic deficit should not be considered as a factor that will determine not to offer therapy in this disease. In most patients it is not due to spinal cord ischemia but to intense peripheral ischemia after acute arterial occlusion.

Regarding fasciotomy, we have become more sensitive to it now in aortic occlusion. We found that younger patients and those with hypercoagulable state were in fact less tolerant to acute aortic occlusion rather than the older patients with preexisting disease. More likely, however, the explanation is that the older cohort of patients already had collateral vessels, and so they tolerated the aortic occlusion better than the young patients who had no established collateral vessels. Furthermore, the younger patients who had a hypercoagulable state also had clotting of their collateral vessels, making them the patients most difficult to treat. To answer your question directly, the fasciotomy is done only when we see that the leg compartment swells after restoration of circulation, or when we have a high index of suspicion.

If we do not restore pulses after aortic thrombectomy or axillofemoral bypass, we obtain an intraoperative angiogram to make sure that there are no distal thrombi or emboli that can be completely missed in the initial angiogram.

Dr. Glen R. Rhodes (Fairfax, Va.). I reported the first patient with heparin-induced thrombocytopenia in 1973. My sense in watching this over a period of time is that, other than trying to exceed some kind of controlled thrombolytic state, either in the patient who has been recognized to have development of the platelet drop, or the patient who had development of thrombosis, there really is no way out for this type of problem. The Canadians in fact have had a lot of experience in trying to give thrombolysis to patients who had development of a platelet drop without thrombosis just to get them out of the situation because there is no lead way in terms of stopping administration of heparin once the platelet count starts to drop. Medicolegally you have to stop administration of heparin. Therefore, in the patient who has not had development of thrombosis, if you can give urokinase or some other lytic agent, it may allow a transition to warfarin (Coumadin) to be able to manage that potential hypercoagulability once you stop the heparin. If you already had thrombosis, you can try a variety of things, including dextran, but you will eventually have to administer Coumadin when you need a transition. I think we are developing an increasing ability to manage urokinase during operation; but I think we are going to develop new techniques for urokinase or some other kind of thrombolytic agent during operation in patients with drains, with reoperations, to try to determine whether we can achieve survival. But I see no other way to try to get a transition with a fibrolytic agent or some other antihypercoagulable agent to be able to have any increased survival rate.